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DOI: [10.1159/000336243](https://doi.org/10.1159/000336243)**Takayasu Arteritis Presenting with Internal Carotid Artery Dissection**R. Galdes^a, P. Batista^a, L.M. Pedro^b, A. Fernandes^c, T.P. Melo^a^aStroke Unit, Department of Neurosciences (Neurology), Departments of ^bVascular Surgery and ^cPathology, Hospital de Santa Maria, Lisbon, Portugal*Background*

In Takayasu arteritis (TA), vessel wall thickening and stenosis are the most common findings, whereas aneurysmal dilatations occur less frequently [1, 2] and arterial dissection is rare. Cerebral ischemia may occur in up to 16% of the patients with TA [3] and can be due to several mechanisms such as subclavian steal in the arm, vessel stenosis/occlusion and embolism from the aorta or the heart [4].

Case Report

A 35-year-old woman presented with a left temporal headache while lifting weight followed by dysphasia and right hemiparesis. Observation disclosed dysphasia, right brachiofacial hemiparesis, weak arterial lower limb pulses and normotension in both arms. Brain MRI revealed left middle cerebral (posterior branch) artery acute ischemia. Cervical vessel ultrasonography and digital subtraction angiography (DSA; fig. 1a–c) showed a left internal carotid artery (ICA) dissection with no other abnormalities. Six months later, the patient had a slight anomia and hemiparesis but complained of transient lower limb claudication. DSA (fig. 1d) and angio-MRI disclosed normal aorta and bilateral external iliac artery occlusion with compensatory circulation and presence of flow at the origin of the femoral arteries, without surgical indication. Initial anticoagulation was substituted by aspirin. A 2-year follow-up cervical vessel ultrasonography showed a concentric segmental right common carotid artery (CCA) 90% stenosis (fig. 1e) and persistent left ICA occlusion. At this point, there were no new symptoms but there was a decrease in the right brachial artery pulse and a blood pressure difference between the arms of 30 mm Hg. Steroids were started but DSA showed persistence of right CCA stenosis (fig. 1f), left ICA occlusion and a new right subclavian artery segmental stenosis (fig. 1g). Prosthetic replacement of the right CCA was performed. Histology of the removed vessel segment showed a panarteritis (fig. 1h–l). Fluorodeoxyglucose PET scan was normal. Cell blood count, erythrocyte sedimentation rate, C-reactive protein, biochemistry, antinuclear, anti-double-stranded DNA and antiphospholipid antibodies, HIV, hepatitis B and C, and Venereal Disease Research Laboratory serology were normal at presentation and in all follow-up

evaluations. Transesophageal echocardiogram and electrocardiogram were unremarkable.

After surgery, azathioprine was added, steroids were tapered to 5 mg and aspirin continued. At 2-year follow-up after CCA surgery, she has been clinically and imagiologically stable.

Discussion

Our patient fulfills all the American College of Rheumatology criteria for TA [5], and vessel vasculitis was histologically proven. Arterial dissection in TA is rare [2, 6], with few reports of aortic dissection [7] and only one report of ICA dissection [8].

In our patient, at the moment of ICA dissection, all the other aortic arches and their branches were angiographically normal and the laboratory tests unremarkable. Extension of arteritis in TA seems to occur in a symmetric way to the paired vascular beds and contiguously in the aortic arch, brachiocephalic trunk and the aorta [1]. Iliac occlusion is an uncommon complication of TA [2, 3]. Our case, with initial ICA and external iliac artery involvement, is atypical but clearly evolved to a typical TA. Large-vessel vasculitis is not in the differentials of ICA dissection [9]; however, ICA dissection can be the first manifestation of TA and is another possible mechanism of cerebrovascular ischemia in TA.

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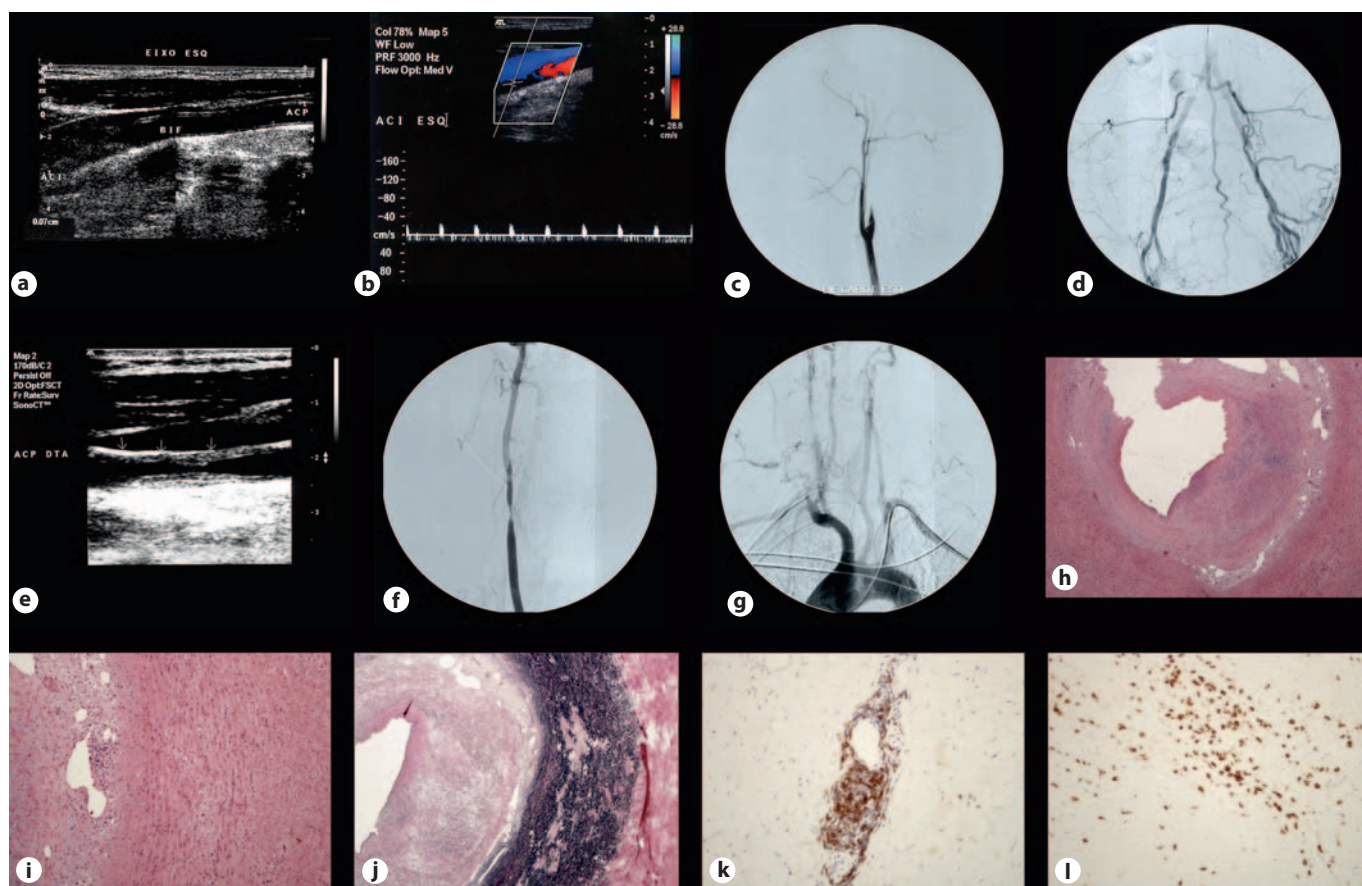


Fig. 1. ICA dissection in TA. Cervical ultrasonography at initial stroke investigation showed the typical signs of left ICA dissection with occlusion, without any wall thickening or inflammatory halo suggesting vasculitis at the left ICA (**a**, **b**) or the contralateral ICA (not shown). The first DSA showing a dissection of the left ICA (**c**) and the lower limb DSA showing an occlusion of the external iliac arteries with compensatory anastomosis to the femoral arteries (**d**). Two years after stroke, cervical ultrasonography showed a concentrically segmental stenosis of the right CCA (**e**), also depicted in DSA 1 month after high-dose steroids (**f**). DSA showed persistent left ICA occlusion and a new right

subclavian artery segmental stenosis 3 cm from the vertebral artery orifice (**g**). Histological examination (hematoxylin-eosin staining) of the removed CCA segment showed a panarteritis: inflammatory cell infiltration, vessel wall thickening with fibrinoid proliferation and neovascularization (**h**), given in detail in a higher magnification (**i**) and with Verhoeff-Van Gieson staining (**j**), showing elastic fiber disruption with a 'moth-eaten' appearance of the media. Immunohistochemistry with anti-CD20 (**k**) and anti-CD3 (**l**) antibodies showed B- and T-lymphocyte infiltration of the three vessel layers, respectively; no giant cells were observed.